

Acid base changes in arterial and central venous blood during cardiopulmonary resuscitation

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SUMMARY

Twenty-seven patients in cardiopulmonary arrest had simultaneous measurements of arterial and central venous blood gases during cardiopulmonary resuscitation (CPR) with a pneumatic chest compression and ventilation device. Mean central venous and arterial hydrogen ion concentrations, P_{CO_2} and calculated bicarbonate concentrations were significantly different ($P < 0.01$) at all sampling times (0, 10 and 20 min). Central venous blood samples predominantly showed a respiratory acidosis in contrast to a mixed disturbance in arterial samples inclined towards a metabolic acidosis.

The mean difference between central venous P_{CO_2} (pcv CO_2) and arterial P_{CO_2} (pa CO_2) ranged from 5.18 to 5.83 kPa reflecting the low blood flow in patients undergoing CPR. Measurement of arterial P_{O_2} indicated adequate oxygenation using the pneumatic device.

Arterial blood gas analysis alone does not reflect tissue acid base status. Bicarbonate administration during CPR may have adverse effects and any decision as to its use should be based on central venous blood gas estimations.

INTRODUCTION

During cardiopulmonary arrest and resuscitation, reduction in tissue perfusion and failure to maintain oxygen delivery result in anaerobic metabolism and lactic acidosis. Measurements of arterial blood gases may reflect, in addition to the lactic acidosis, the effects of ventilation or the metabolic and respiratory derangements

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preceding the arrest. However, in animal studies of CPR, during which mixed venous and arterial blood gases were measured, Grundler *et al.* (1986) concluded that mixed venous blood sampling more accurately reflected tissue acid-base status. Despite studies of CPR in critically ill patients confirming these observations (Weil *et al.*, 1986), the use of arterial blood gas measurements for assessing the adequacy of ventilation and the need for bicarbonate therapy remains widespread (Resuscitation Council, 1984).

Clearly, caution is necessary before extrapolating from studies carried out in a critical care environment to the more common occurrence of sudden and unexpected cardiac arrest. The aim of this study, therefore, was to determine the patterns of arterial and central venous blood gas measurements in patients undergoing CPR in an A&E department.

SUBJECTS AND METHODS

Adults with non-traumatic, normothermic cardiac arrest, admitted to the A&E Department of Edinburgh Royal Infirmary were studied. All patients had sustained cardiorespiratory arrest in the pre-hospital setting and had received Basic Life Support prior to A&E Department admission.

On admission patients were treated according to the guidelines of the U.K. Resuscitation Council (1984). Electrical DC counter-shock and anti-arrhythmic agents were used as appropriate. All patients were intubated and ventilated with 100% oxygen and mechanical chest compression was performed using a Michigan Instruments Thumper, model 1004 (Michigan Instruments, Grand Rapids, MI, U.S.A.), functioning at a ratio of five chest compressions to one ventilation (Little *et al.*, 1974). CPR was performed with sufficient force to compress the chest 2.5–3" (80–100 lbs) and ventilation was set at 30 cm H₂O. A 20 cm radio-opaque single lumen catheter (long line – Surcath, Vygon (U.K.) Ltd., Cirencester, U.K.) was inserted percutaneously into the right subclavian vein and advanced into the right atrium. Correct positioning of the catheter was verified in all patients by chest X-ray following the resuscitation attempt.

On admission, at the commencement of Advanced Cardiac Life Support, blood samples were withdrawn simultaneously from the central venous line and the femoral artery (0 time). Subsequently samples were taken at 10 and 20 min during resuscitation. Blood samples were considered consistent with arterial sampling when P_{O_2} exceeded 6.7 kPa (5). Statistical analysis was performed using a paired *t*-test. Data are reported as means (SEM) and differences considered significant when $P < 0.05$.

RESULTS

Twenty-seven patients were prospectively studied. The group comprised 20 men and seven women ranging from 26 to 84 years of age (median 65 years). The time

between onset of cardiac arrest and arrival in the A&E Department averaged 15 min. The presenting arrhythmia was asystole in 16 patients, ventricular fibrillation in seven patients and four patients had electromechanical dissociation.

Paired central venous and arterial samples were available for analysis in 19, 20 and 14 patients at 0, 10 and 20 min respectively. Sixteen patients received 8.4% sodium bicarbonate at a mean dose of 77 mEq between the 2 and 10-min times (mean 7.3 min) as dictated by clinical circumstances and arterial blood gas analysis.

H^+ concentrations in simultaneous arterial and central venous blood samples taken serially were all elevated but there were large and statistically significant differences between the two groups (Fig. 1). The proportion of patients with low or normal H^+ concentration on arterial sampling were 6/19 (32%) at the 0 time, 5/20 (20%) at 10 min and 2/14 (14%) at 20 min. All central venous samples, however, revealed acidaemia.

A statistically significant difference was demonstrated between $Paco_2$ and $Pcvco_2$ (Fig. 2). However, the mean $Paco_2$ during resuscitation fell within the normal range (4.8–6.0 kPa). The proportion of patients demonstrating hypo or normocapnoea on arterial sampling were 13/19 (68%) at the 0 time, 13/20 (65%) at 10 min and 7/14 (50%) at 20 min. In contrast the values for patients with hypercapnoea on central venous sampling were 26/27 (96%), 24/24 (100%) and 12/16 (88%) respectively. The mean difference in arterial/central venous Pco_2 was noted to increase between the 0 and 10-min times, but this was not statistically significant.

The mean calculated bicarbonate concentrations in arterial blood did not change significantly during resuscitation. Although the mean bicarbonate concentration in central venous blood rose from 20 to 24 mmol l^{-1} between the 0 and 10-min times, this was not significant. The mean arterial/central venous differences in calculated bicarbonate were, however, very significantly different (Fig. 3).

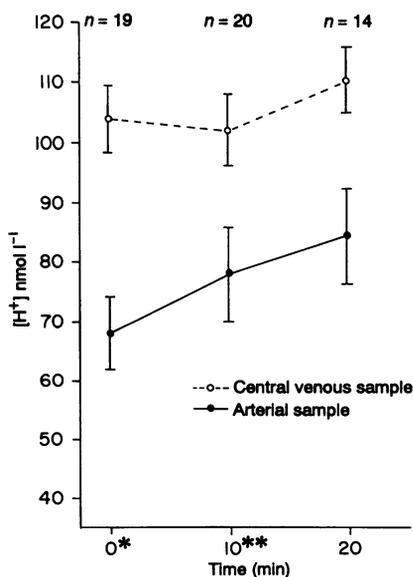


Fig. 1. Central venous and arterial H^+ concentrations (nmol l^{-1}). * $P < 0.0001$, ** $P < 0.01$.

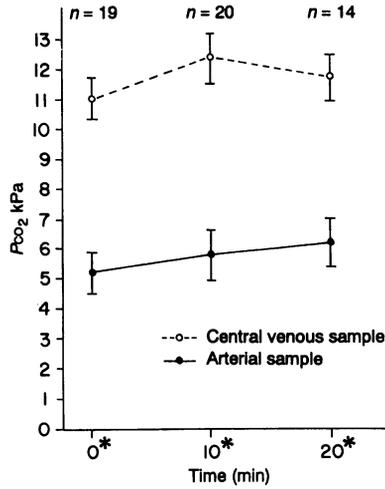


Fig. 2. Central venous and arterial PCO_2 (kPa). * $P < 0.0001$.

Measurements of PO_2 indicated adequate arterial oxygenation during resuscitation and there were large and significant arterial/central venous PO_2 differences (Fig. 4). No patient survived the resuscitative effort.

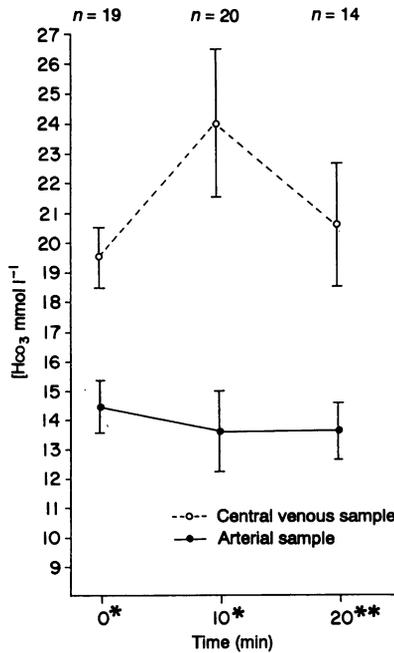


Fig. 3. Central venous and arterial calculated bicarbonate concentrations ($mmol\ l^{-1}$). * $P < 0.0001$, ** $P < 0.01$.

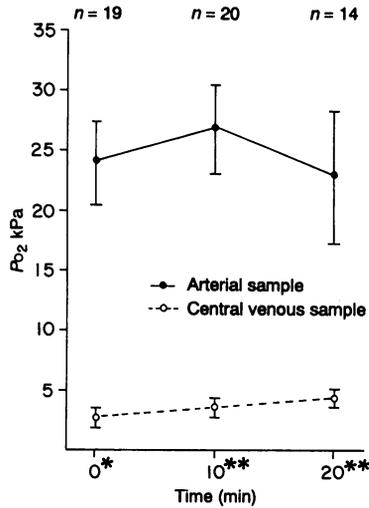


Fig. 4. Central venous and arterial P_{O_2} (kPa). * $P < 0.0001$, ** $P < 0.01$.

DISCUSSION

Recent studies have shown that respiratory acidosis appears selectively in mixed venous blood during CPR (Grundler *et al.*, 1986; Weil *et al.*, 1986). Weil *et al.*, (1986) reported on differences in acid-base status between mixed venous and arterial blood (median sampling time, 23 min) in 16 critically ill patients all of whom were intubated and receiving mechanical ventilation prior to cardiac arrest. During CPR with manual external cardiac compression, and variable amounts of administered bicarbonate, the arterial pH averaged 7.41 (nine patients were alkalaemic) whereas the mean pH in mixed venous blood was 7.15. The mean arterial pressure MAP of CO_2 was 32 mmHg in contrast to a mean mixed venous P_{CO_2} of 74 mmHg. There was no significant difference in calculated concentration of bicarbonate which averaged 25 mmol l^{-1} in arterial blood, and 23 mmol l^{-1} in mixed venous blood.

In this study in patients with sudden cardiac arrest, both mean central venous and arterial blood specimens were acidemic throughout the sampling period. A severe respiratory acidosis predominated in central venous blood, whereas a metabolic acidosis was present in arterial blood. A similar acid-base pattern was reported by Nowak *et al.* (1987) in 35 patients with pre-hospital cardiac arrest (mean time prior to arrival in A&E, 15 min), when single central venous and arterial samples were taken at an average time of 16 min following admission. The observation that in prolonged cardiopulmonary arrest, acidosis appears in both arterial and mixed venous blood, has been demonstrated in previous studies (Bishop & Weisfeldt, 1976; Grundler *et al.*, 1986).

The striking hypercapnoea in venous blood is attributed to decreased pulmonary excretion of CO_2 following abrupt reduction in pulmonary blood flow during CPR (Weil *et al.*, 1986). Measurements of end-tidal expired CO_2 have been shown to fall

to 35% of pre-arrest levels (Falk *et al.*, 1988). The marked decrease in pulmonary blood flow results in a large increase in the ventilation/perfusion ratio and accounts for the mean arterial P_{CO_2} falling within normal limits during resuscitation (Weil *et al.*, 1986). The severe respiratory acidosis observed in venous blood has potentially important physiological and therapeutic implications. CO_2 can rapidly diffuse across cell membranes, potentially lowering intra-cellular pH (Weisfeldt *et al.*, 1975). In isolated muscle preparations, myocardial contractility is more profoundly affected by increases in P_{CO_2} than by decreases in extra-cellular pH (Poole-Wilson & Langer, 1975; Poole-Wilson, 1975). Bicarbonate administration results in the generation of carbon dioxide which may aggravate the intra-cellular acidosis occurring during CPR. This situation will be compounded if bicarbonate therapy is guided solely by arterial blood gas measurements, and the severe venous respiratory acidosis is not appreciated. The respiratory acidosis represents an important component of tissue acidemia during CPR and central venous measurements may therefore reflect a more accurate picture of tissue acid-base status. The proximal venous and tissue CO_2 levels are probably even higher than measured $P_{cv}CO_2$ levels (Kinney, 1960).

$P_{cv}CO_2$ represents a balance between CO_2 production from tissue metabolism and carbon dioxide elimination (Powles & Campbell, 1978). During cardiopulmonary arrest, CO_2 production results from residual aerobic metabolism and subsequently from the buffering of lactic acid generated from anaerobic metabolism (Hodgekins *et al.*, 1981). Central venous P_{CO_2} is inversely related to both cardiac output and alveolar ventilation. Changes in alveolar ventilation cause similar alterations in P_{aco_2} and $P_{cv}CO_2$ whereas a decrease in cardiac output will cause an increase in the difference between these two measurements (Powles & Campbell, 1978). The large arterial/central venous differences in P_{CO_2} that we report, are probably due to the poor cardiac output achieved during CPR. These differences were, however, less than those achieved during cardiopulmonary resuscitation by closed chest compression in a comparable group of pre-hospital cardiac arrest patients treated in North America (mean $P_{cv}-P_{aco_2} = 60.5$ mmHg) (Grundler *et al.*, 1986), but less than that achieved during open chest CPR in a canine model (mean $P_{cv}-P_{aco_2} = 15.51$ mmHg) (Martin *et al.*, 1985). The mean $P_{cv}-P_{aco_2}$ differences noted in the present study, fell between the 0 time and 20 min. This may reflect an improvement in blood flow but was not statistically significant. It does, however, suggest that the mechanical device is more efficient than the often erratic and poorly performed manual technique (Skinner *et al.*, 1985).

Measurements of arterial PO_2 in this study indicate adequate oxygenation using the mechanical device. The problem of differentiating arterial from venous blood during percutaneous femoral sampling in cardiac arrest studies has been previously highlighted (Henneman *et al.*, 1988). In addition, standard CPR has been shown to produce a pressure pulse in the aorta and inferior vena cava of comparable magnitude (Niemann *et al.*, 1984) and the femoral arterial pulsation is often less than the femoral venous one due to atherosclerotic disease (Henneman *et al.*, 1988). While a low PO_2 is suggestive of venous sampling, pulmonary congestion or inadequate pulmonary perfusion can significantly limit oxygenation during CPR (Fillmore *et al.*, 1970). A PO_2 greater than 50 mmHg at an elevation of 581 ft (Detroit) has previously been used as evidence of arterial sampling from patients

undergoing CPR (5). The large arterial/central venous PO_2 differences we report, confirm that intermittent percutaneous femoral sampling can be satisfactorily used for study purposes allowing differentiation of arterial from venous blood on the basis of PO_2 .

We have previously reported our success rates for cardiopulmonary resuscitation in sudden cardiac arrest (Robertson & Little, 1984). As with other reports, recovery rates following asystole are very poor (<5%). The fact that, in this study, no patient survived the resuscitative attempt reflects this, together with the fact that successful resuscitation for patients with ventricular fibrillation persisting for 10 min or more is also very uncommon.

This study highlights striking differences in the acid-base conditions of arterial and central venous blood during CPR. Analysis of arterial blood gases reveals a mixed disturbance inclined towards a mild metabolic acidosis, whereas simultaneous analysis of central venous blood reveals a predominant and profound respiratory acidosis. Arterial sampling is necessary to monitor the oxygenation of blood being delivered to the tissues. Administration of sodium bicarbonate during cardiac arrest may be counter-productive since it may increase tissue and central venous blood carbon dioxide tensions. The decision as to its administration should therefore be based on blood gas analysis of central venous blood.

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